A Biological Model for Delayed Recall of Childhood Abuse

J. Douglas Bremner

SUMMARY. There is currently scientific controversy related to the validity of delayed recall of memories of childhood abuse. Post-Traumatic Stress Disorder (PTSD), which affects 8% of the general population, is a possible consequence of childhood abuse. Changes in brain structures and systems mediating memory offer a possible explanation for delayed recall of childhood abuse in patients with abuse-related PTSD. Brain areas affected by traumatic stress are involved in memory and the modulation of emotion and include the hippocampus and medial prefrontal cortex. Stress also results in acute and chronic changes in neurochemical systems, including cortisol and norepinephrine, that strengthens or weakens the laying down of memory traces. Patients with PTSD have alterations in a broad range of memory functions, including insertions, deletions and distortions. PTSD patients also show changes in structure and function in brain regions mediating memory, including the hippocampus and medial prefrontal cortex, as well as in brain chemical systems involved in the stress response that influence the laying down and retrieval of memories, including cortisol and nore-

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pinephrine. The effects of stress on the brain highlight the importance of considering PTSD in research on memory that is generalized to questions about the delayed recall of childhood abuse.

KEYWORDS. Trauma, memory, hippocampus, neurobiology, PTSD

INTRODUCTION

Delayed recall of childhood abuse is a controversial topic that has had a major impact on our society. Self-reported childhood sexual abuse affects 16% of women (about 40 million) in this country (including rape, attempted rape, or molestation) at some time before their 18th birthday (McCauley et al., 1997) and one million new cases of childhood abuse are documented each year. Post-Traumatic Stress Disorder (PTSD), a psychiatric disorder associated with exposure to psychological traumas, such as childhood abuse, is characterized by symptoms including intrusive memories, hyperarousal, sleep disturbance, flashbacks, increased emotional responsivity, dissociation, and problems with memory and concentration (Saigh & Bremner, 1999). PTSD affects 8% of the general population at some time in their lives (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), and is seen in about 15% of individuals exposed to a severe psychological trauma, such as childhood sexual abuse.

Alterations in memory form an important part of the clinical presentation of patients with childhood abuse-related PTSD (Bremner & Marmar, 1998). As reviewed in this article, PTSD patients report deficits in declarative memory (memory for facts or lists, as reviewed below), fragmentation of memories (both autobiographical and trauma-related), memory distortions, and dissociative amnesia (gaps in memory that can occur for minutes to days and are not due to ordinary forgetting). We have demonstrated significant increases in dissociative amnesia based on structured interviews in combat veterans with PTSD compared to combat veterans without PTSD (Bremner, Steinberg, Southwick, Johnson, & Charney, 1993). Many abuse victims claim to remember only certain aspects of the abuse event, a phenomenon related to dissociative amnesia. The wide range of effects that traumatic stress has on memory complicates questions related to delayed recall of childhood abuse.

There is a limited amount of research on delayed recall of childhood abuse. Cases of individuals who have no memory for childhood sexual abuse

and then suddenly remember an abuse event years after the fact have been widely publicized in the popular literature. For example, one man who was listening to a report of a priest who was arrested for molesting children 20 years ago suddenly had a memory of being molested by that particular priest. The validity of this type of delayed recall of childhood abuse has been hotly debated in the scientific literature, as reviewed elsewhere in this volume. Studies show that memories are, in fact, susceptible to insertions, deletions, and distortions, often resulting in a situation where the individual remains convinced of the validity of the memory as experienced in its altered form (Kihlstrom, 1987). A variety of experimental paradigms have demonstrated the capacity for false recall, as reviewed in detail elsewhere in this volume. Findings from these studies have led to criticism of clinical reports of delayed recall of childhood abuse. The clinical and scientific literature related to delayed recall of childhood abuse has become polarized with two conflicting viewpoints related to whether or not delayed recall of childhood abuse are valid.

In the current article, I outline a model for how changes in the brain may mediate delayed recall of childhood abuse. One of the primary criticisms of the viewpoint that delayed recall represents valid memories is the apparent illogicality of forgetting of events that most people would consider impossible to forget. However, studies in animals have demonstrated that stress can impair memory function in some circumstances, acting through stress hormones and brain chemicals that affect the way memories are laid down. In addition, stress can result in lasting changes in the structure and function of brain areas involved in memory. The results of animal studies of stress have been used to direct hypotheses related to the effects of stress on patients with abuse-related PTSD. Many of the findings from animal studies have been replicated in humans, including findings of long-term alterations in brain systems and structures involved in the stress response and memory (Bremner, Southwick, & Charney, 1999). Good research data on the proportion of PTSD patients who experience delayed recall of abuse does not exist; however, clinical experience dictates that fragmentations and alterations in memory for traumatic events are more common in patients with PTSD, and include memories for some events that are continuous, memories for other events that are delayed or fragmented, and other memories that are never retrieved at all.

HIPPOCAMPAL DYSFUNCTION AS A POTENTIAL CAUSE OF DELAYED RECALL OF ABUSE

Stress-induced hippocampal damage represents one possible mechanism for delayed recall of childhood abuse. The hippocampus is a brain area involved in learning and memory (Squire & Zola-Morgan, 1991) that is particularly sensitive to stress (Bremner, 1999; McEwen et al., 1992; Sapol-

sky, 1996). This function is critical to the stress response, for example, in assessing potential threat during a life-threatening situation, as occurs with exposure to a predator. Work from laboratories such as Sapolsky (1996) at Stanford University and McEwen et al. (1992) at Rockefeller University demonstrated that, in a variety of animal species, high levels of glucocorticoids (cortisol in man) seen in stress are associated with damage to the hippocampus. When male and female vervet monkeys are caged together, the female monkeys attack the males, leading to extreme stress in the males which is often fatal. Monkeys who were improperly caged and died spontaneously following exposure to severe stress had multiple gastric ulcers on autopsy, consistent with exposure to chronic stress, as well as enlarged adrenal cortices, consistent with sustained glucocorticoid release. Stress was found to result in damage to the CA3 subfield of the hippocampus (Uno, Tarara, Else, Suleman, & Sapolsky, 1989) that was related to exposure to glucocorticoids (Sapolsky, Uno, Rebert, & Finch, 1990). Studies showed that direct glucocorticoid exposure to the hippocampus results in decreased dendritic branching (Woolley, Gould, & McEwen, 1990), a loss of neurons, and an inhibition of neuronal regeneration (Gould, Tanapat, McEwen, Flugge, & Fuchs, 1998). Glucocorticoids disrupt cellular metabolism (Lawrence & Sapolsky 1994), thereby increasing the vulnerability of hippocampal neurons to excitatory amino acids like glutamate (Virgin et al., 1991). Other neurochemical systems interact with glucocorticoids to mediate the effects of stress on memory and the hippocampus, including brain-derived neurotrophic factor (BDNF) (Nibuya, Morinobu, & Duman, 1995; Smith, Makino, Kvetnansky, & Post, 1995).

Stress also has effects on functions of new learning and memory that are mediated by the hippocampus. Exposure to the stress of an unfamiliar environment resulted in deficits in working memory indicative of hippocampal dysfunction (Diamond, Fleshner, Ingersoll, & Rose, 1996), while high levels of glucocorticoids seen with stress were associated with both deficits in new learning and damage to the hippocampus (Luine, Villages, Martinex, & McEwen, 1994). Long-term subcutaneous implants of glucocorticoids that mimic the chronic stress situation resulted in deficits in new learning and memory for maze-escape behaviors (Arbel, Kadar, Silberman, & Levy, 1994). Stress was also shown to affect long-term potentiation (LTP), which is used as a model for the molecular basis of new learning and memory (Diamond, Branch, Fleshner, & Rose, 1995).

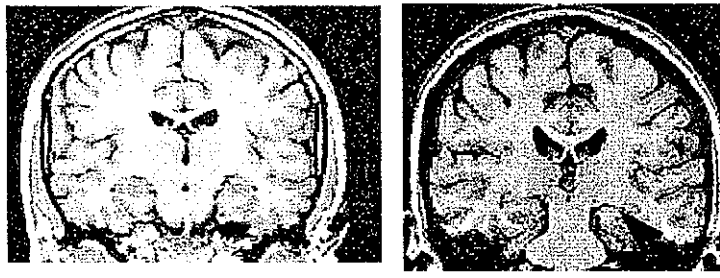
Stress-mediated hippocampal damage may lead to dysregulation of others aspects of the organism's stress response system. The hippocampus has an inhibitory effect on the corticotropin releasing factor (CRF)/(hypothalamic-pituitary-adrenal (HPA) axis (Jacobson & Sapolsky, 1991). CRF, which is released during stress, has behavioral effects that are characteristic of anxiety or the stress response. Stress-induced damage to the hippocampus results in

increased levels of CRF (Herman et al., 1984). Consistent with this, an increase in CRF is seen in patients with PTSD, as measured in cerebrospinal fluid (Bremner, Licinio et al., 1997).

In order to test hypotheses about the effects of traumatic stress on memory function in human subjects based on the animal studies reviewed above, we used neuropsychological testing to measure declarative memory function in PTSD. We selected measures that were validated in studies of patients with epilepsy to be specific probes of hippocampal function. Sass and colleagues (1990) in the Yale Neurosurgery Program administered the Wechsler Memory Scale (WMS)-Logical Subscale (paragraph recall) (Russell, 1978) and verbal Selective Reminding Test (vSRT) (Hannay & Levin, 1985) to patients with epilepsy who subsequently underwent surgical resection of the hippocampus. These investigators found that decreases in percent retention of the WMS paragraph after delayed recall, and deficits on the Long Term Retrieval (LTR) subscale of the vSRT were correlated with decreases in neuronal number of the CA3 region of the left hippocampus. The findings were specific to verbal, and not visual, memory. In an initial study, we (Bremner, Scott et al., 1993) found deficits in verbal declarative memory function in combat-related PTSD. These declarative memory deficits included problems with paragraph recall, as measured by the WMS, for immediate and delayed recall, and percent retention of the paragraph on delay. Patients also had problems with new learning of word lists, as measured by the vSRT. IQ and visual memory were intact. In order to test the hypothesis that traumatic stress results in hippocampal damage, we (Bremner, Randall, Scott et al., 1995) used magnetic resonance imaging (MRI) to quantitate hippocampal volume in living human subjects with a history of traumatic stress and a diagnosis of PTSD. An initial study showed an 8% smaller right hippocampal volume (but not comparison regions) in Vietnam veterans with combat-related PTSD (Figure 1). Decreases in right hippocampal volume in the PTSD patients were associated with deficits in short-term memory, as measured by the WMS-Logical, percent retention subcomponent ($r = .64$, $p < .05$).

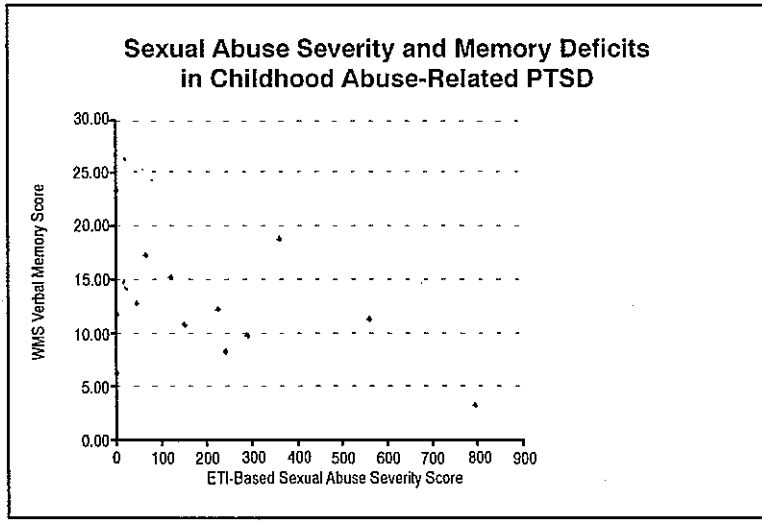
Next, we (Bremner, Randall, Capelli et al., 1995) looked at survivors of childhood physical and/or sexual abuse with a diagnosis of PTSD (n = 18), and compared them to healthy subjects (n = 17) matched for age, sex, race, years of education, and years of alcohol abuse. We found deficits in the ability to recall a paragraph, both immediately and after a 30 minute delay, as measured by the WMS, in patients with abuse-related PTSD. These patients also showed deficits in the ability to learn new words, as measured by the SRT ($p < .01$). Deficits in short-term memory in the childhood abuse patients were significantly correlated with level of abuse, as measured by the composite severity score on the Early Trauma Inventory (Bremner, Vermetten, & Mazure, in press) ($r = -.48$, $p < .05$) (Figure 2). There was no difference in

FIGURE 1. Magnetic Resonance Imaging (MRI) Scan of the Hippocampus in a Normal Control and a Patient with PTSD. There Is a Visible Reduction in Hippocampal Volume in the PTSD Patient



NORMAL PTSD

FIGURE 2. Deficits in Verbal Declarative Memory Were Correlated with Sexual Abuse Severity in Patients with Abuse-Related PTSD ($r = .48$, $p < .05$)



IQ or visual memory. Using MRI we (Bremner, Randall et al., 1997) measured hippocampal volume in 17 male and female adults with a history of severe childhood physical and/or sexual abuse and long-term psychiatric consequences in the form of PTSD, and compared them to 17 case-matched healthy controls. There was a 12% reduction in left hippocampal volume in the patients with abuse-related PTSD in relation to comparison subjects. This reduction was statistically significant ($p < .05$). A 3.8% reduction in volume of the right hippocampus was not significant.

Other studies found reductions in hippocampal volume in clinical populations of traumatized subjects. Stein, Koverola, Hanna, Torchia, and McClarty (1997) found a statistically significant 5% reduction in left hippocampal volume in 21 sexually abused women, relative to 21 nonabused female controls. Most (although not all) of the abused women had a current diagnosis of PTSD. Gurvits et al. (1996) compared hippocampal volume in seven patients with Vietnam combat-related PTSD to seven Vietnam combat veterans without PTSD, and eight healthy nonveteran controls. The authors found a 26% bilateral decrease in hippocampal volume, which was statistically significant for both left and right hippocampal volume considered separately. Although subjects were not case matched for alcohol abuse, there continued to be a significant difference in hippocampal volume after adjusting for years of alcohol abuse using analysis of covariance. In summary, there are several replicated studies in more than one population of traumatized patients showing atrophy of the hippocampus, which appears to be specific to a PTSD diagnosis.

Declarative memory deficits have an important effect on PTSD patients. These patients cannot remember simple things on a daily basis and, for this reason, are often unable to hold regular jobs. The magnitude of memory impairment is a 40% reduction on neuropsychological tests relative to normal controls and is equivalent to patients with epilepsy who have had their hippocampus surgically resected (Lencz et al., 1992). My patients often describe the debilitating effects of their memory impairments on their social and occupational functioning. For example, one of my abuse-related PTSD patients complained that she felt like she had an early dementia. I frequently counsel my patients not to pursue a career or training that requires memorization.

Empirical studies on memory and the hippocampus may shed some light on the controversy surrounding delayed recall of memories of childhood abuse. The hippocampus plays an important role in integrating or binding together different aspects of a memory at the time of recollection (Squire & Zola-Morgan, 1991). It is felt to be responsible for locating the memory of an event in time, place, and context. We (Bremner, Krystal, Charney, & Southwick, 1996) have hypothesized that atrophy and dysfunction of the hippocampus following exposure to childhood abuse may lead to distortion and fragmentation of memories.

For example, an abused patient who was locked in the closet remembered the smell of old clothes, but had no visual memory of being in the closet, and no affective memory of fear. Perhaps with psychotherapy there is a facilitation of associations to related events that may bring all of the aspects of the memory together. Or, if the patient experiences an event such as being trapped in a dark elevator, the feeling of fear associated with darkness and the enclosed space may be enough to trigger a recollection of the entire memory.

NEUROCHEMICAL MODULATION OF MEMORY

Animal studies of the effects of stress early in development on neurochemical systems have implications for delayed recall of childhood abuse. The cortisol hormonal system is particularly relevant because cortisol released during stress acts over a period of hours to weaken the strength of memory traces laid down at the time of a stressful event (reviewed in De Wied & Croiset, 1991). Stress is associated with an acute increase in cortisol in humans (Rose, Poe, & Mason, 1968). Animal models of childhood abuse and neglect demonstrated that animals exposed to stress early in life subsequently have an increased sensitivity of glucocorticoid responsiveness throughout their lifespan (Fride, Dan, Feldon, Halevy, & Weinstock, 1986; Ladd, Owens, & Nemeroff, 1996; Levine, 1962; Levine, Weiner, & Coe, 1993; Plotsky & Meaney, 1993; Stanton, Gutierrez, & Levine, 1988). PTSD is associated with chronic dysregulation of the cortisol system. The findings are most consistent with elevated levels of cortisol in children in the early aftermath of childhood abuse (Lemieux & Coe, 1995), which may lead to chronic dysregulation and low cortisol levels in adults with chronic PTSD many years after the psychological trauma (Yehuda, Southwick, Nussbaum, Giller, & Mason, 1991). These findings are relevant to the delayed recall controversy, because changes in cortisol levels in childhood abuse survivors will affect how memories are laid down and retrieved.

The adrenaline system (norepinephrine) is another neurochemical system that plays a critical role in the stress response that is relevant to delayed recall of childhood abuse (reviewed in Bremner, Krystal, Southwick et al., 1996). Norepinephrine released during stress has a short period of action to strengthen the laying down of memory traces (reviewed in McGaugh, 1989). Childhood abuse-related PTSD is associated with increased levels of norepinephrine, as measured in 24-hour urine (Lemieux & Coe, 1995). Stimulation of the norepinephrine system with a medication called yohimbine (a noradrenergic alpha-2 receptor antagonist) results in increased symptoms of PTSD in patients with combat-related PTSD, suggesting that increased norepinephrine activity underlies symptoms of PTSD (Southwick et al., 1993). Patients with combat-related PTSD administered yohimbine had differences in brain function compared to

healthy subjects that were also consistent with increased norepinephrine release in the brain following administration of this drug (Bremner, Innis et al., 1997). These findings are consistent with long-term alterations in memory function in PTSD. Alterations in noradrenergic activity in PTSD may lead to changes in memory encoding and/or retrieval.

Modulation of memory function by cortisol and norepinephrine may represent a mechanism of delayed recall of childhood abuse. As mentioned above, cortisol acts over a period of hours to weaken the laying down of memory traces, while norepinephrine has a rapid effect to strengthen memory traces. Long-term dysregulation of these systems may result in chronic changes in the way memories are retrieved in abuse survivors with PTSD. For example, exaggerated cortisol release during stress in PTSD may result in an inhibition of memory retrieval. This may account for the finding that rape victims report that memories for the rape trauma are actually less clear than those for neutral memories (Koss, Figueredo, Bell, Tharan, & Tromp, 1996). Exaggerated release of norepinephrine with stress in PTSD would actually be expected to facilitate recall based on the animal studies cited above. Such a mechanism may be responsible for the sudden eruption into consciousness of long-lost memories of childhood abuse during adult stressors that some PTSD patients claim to experience. Both acute and chronic responses of these neurochemical systems to stress need to be considered in order to understand alterations in memory encoding and retrieval that we propose underlie delayed recall of childhood abuse.

DYSFUNCTION OF THE MEDIAL PREFRONTAL CORTEX AS A MECHANISM OF DELAYED RECALL OF CHILDHOOD ABUSE

Abnormalities of frontal lobe function may also underlie delayed recall of childhood abuse. The medial prefrontal cortex is of particular interest because of the role it plays in emotion, social behavior, and inhibition of responses. Human subjects with lesions of medial prefrontal cortical areas (e.g., the famous case of Phineas Gage; Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994) have deficits in interpretation of emotional situations that are accompanied by impairments in social relatedness. Other aspects of prefrontal cortical function may be relevant to PTSD. The prefrontal cortex is involved in selecting responses and planning for execution of action. Patients with lesions of the prefrontal cortex exhibit a variety of abnormalities of cognition, including impairments in ability to select a correct response, as well as insertion, distortions, and confabulations of memory.

These types of memory alterations are seen in patients with abuse-related PTSD. We (Bremner, Shobe, & Kihlstrom, 2000) used a paradigm to assess

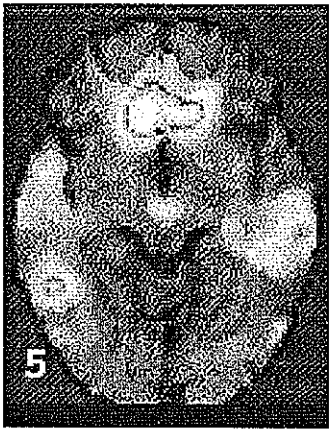
capacity for false memory that assesses free recall using lists of words that are all highly associated with a single primary associate ("critical lure") (Roediger & McDermott, 1995). For example, for the critical lure *needle*, the list words were *thread, pin, eye, sewing, sharp, point, prick, thimble, haystack, thorn, hurt, injection, syringe, cloth, knitting*. During free recall, about 40% of normal subjects falsely recalled the "critical lure" word *needle*. We used this paradigm to assess the propensity for false memory recall in 63 subjects, including women with a self-reported history of early childhood sexual abuse (with and without a diagnosis of PTSD), and healthy men and women without a history of childhood abuse. Women with abuse-related PTSD had a higher frequency of false recognition recall of critical lures (95%) than women with abuse histories without PTSD (76%), and non-abused non-PTSD women (79%). There were no differences between normal men and normal women. PTSD women also showed a pattern of poorer recall of previously studied words, consistent with previous findings of declarative memory deficits in PTSD, and a greater number of intrusions on nonstudied words other than critical lures. These findings are consistent with a greater propensity for distortions in memory in women with self-reported childhood sexual abuse and PTSD. As noted above, prefrontal cortical dysfunction in abuse-related PTSD is a possible explanation for these phenomena, which would explain the increase in capacity for distortion and source amnesia effects.

Medial prefrontal cortical dysfunction in abuse-related PTSD could explain both cognitive deficits (insertions and distortions), as well as problems regulating mood and emotion. Medial prefrontal cortical areas modulate emotional responsiveness through inhibition of amygdala function, and we have hypothesized that dysfunction in these regions may underlie pathological emotional responses in patients with PTSD (Bremner, Narayan et al., 1999). The development of conditioned fear responses, as in the pairing of a neutral stimulus (bright light–the conditioned stimulus) with a fear-inducing stimulus (electric shock–the unconditioned stimulus), which leads to fear responses to the light alone, is mediated by the amygdala (Davis, 1992; LeDoux, 1993). Repeated exposure to the conditioned stimulus alone normally results in the gradual loss of fear responding. The phenomenon, known as extinction to conditioned fear responses, has been hypothesized to be secondary to the formation of new memories that mask the original conditioned fear memory (Bouton & Swartzentruber, 1991). The extinguished memory is rapidly reversible following reexposure to the conditioned-unconditioned stimulus pairing, even up to one year after the original period of fear conditioning (McAllister & McAllister, 1988), suggesting that the fear response did not disappear, but was merely inhibited. This inhibition may take place through connections between the medial prefrontal cortex and the

amygdala (Carmichael & Price, 1995; Devinsky, Morrell, & Vogt, 1995; Vogt, Finch, & Olson, 1992).

Imaging studies of brain function in PTSD are consistent with dysfunction of the medial prefrontal cortex in PTSD. We (Bremner, Innis et al., 1997) stimulated PTSD symptoms with the noradrenergic agent, yohimbine, and found a relative failure of activation in metabolism in parts of the medial prefrontal cortex, as well as decreased function in the hippocampus, as measured by positron emission tomography (PET) assessment of metabolism in combat-related PTSD compared to healthy controls. In a second study of combat-related PTSD, using PET and $[^{15}0]H_20$ measurement of cerebral blood flow, we (Bremner, Staib et al., 1999) studied 10 Vietnam veterans with PTSD and 10 Vietnam veterans without PTSD during exposure to combat-related and neutral slides and sounds. Vietnam veterans with combat-related PTSD demonstrated a decrease in blood flow in the medial prefrontal cortex (Brodmann's area 25, or subcallosal gyrus) and the medial temporal cortex (auditory cortex) during exposure to combat-related slides and sounds. These changes were not seen in the non-PTSD combat veterans. There was also a failure of activation in the anterior cingulate (areas 32 and 24), and increased activation in the posterior cingulate, motor cortex, and lingual gyrus in PTSD (Figure 3). In another study, we (Bremner, Narayan et al., 1999) examined cerebral blood flow correlates of exposure to personalized scripts of childhood sexual abuse in women with histories of childhood abuse, with (n = 10) and without (n = 12) PTSD. PTSD women showed

FIGURE 3. Statistical Parametric Map Overlaid on an MRI Showing Areas of Decreased Blood Flow During Traumatic Reminders in PTSD.



decreased blood flow in the medial prefrontal cortex (area 25), and failure of activation in the anterior cingulate, with increased blood flow in the posterior cingulate and motor cortex (replicating findings in combat-related PTSD), and anterolateral prefrontal cortex. PTSD women also had decreased blood flow in the right hippocampus, parietal and visual association cortex.

Other studies (Rauch et al., 1996; Shin et al., 1997) of traumatic imagery in combat-related PTSD found alterations in the orbitofrontal and temporal cortex in patients with PTSD. These imaging findings are consistent with dysfunction of the medial prefrontal cortex in PTSD. Medial prefrontal cortical/anterior cingulated activation may represent a "normal" brain response to traumatic stimuli that serves to inhibit feelings of fearfulness when there is no true threat. Failure of activation in this area, and/or decreased blood flow in the adjacent medial prefrontal cortex in PTSD, may lead to increased fearfulness that is not appropriate for the context, a behavioral response that is highly characteristic of patients with PTSD. If abuse-related PTSD patients are unable to regulate emotional responses to exposure to cues of the original trauma, this may lead to behaviors in which patients avoid reminders in order to protect themselves, leading to "amnesia" which is only overcome in unusual circumstances that are later identified as "delayed recall." Consistent with this idea, studies do show that PTSD symptoms increase after delayed recall of childhood abuse.

Medial prefrontal cortical dysfunction may also play a role in the increase in intrusions, distortions, and source amnesia seen in patients with PTSD. In addition, considering the role of the medial prefrontal cortex in inhibition of responses, dysfunction in this area may underlie the dysregulation of memory inhibition and access, including childhood abuse memories in PTSD, further facilitating delayed recall of childhood abuse.

RELEVANCE OF THE EFFECTS OF TRAUMATIC STRESS ON MEMORY SYSTEMS TO PSYCHOTHERAPY

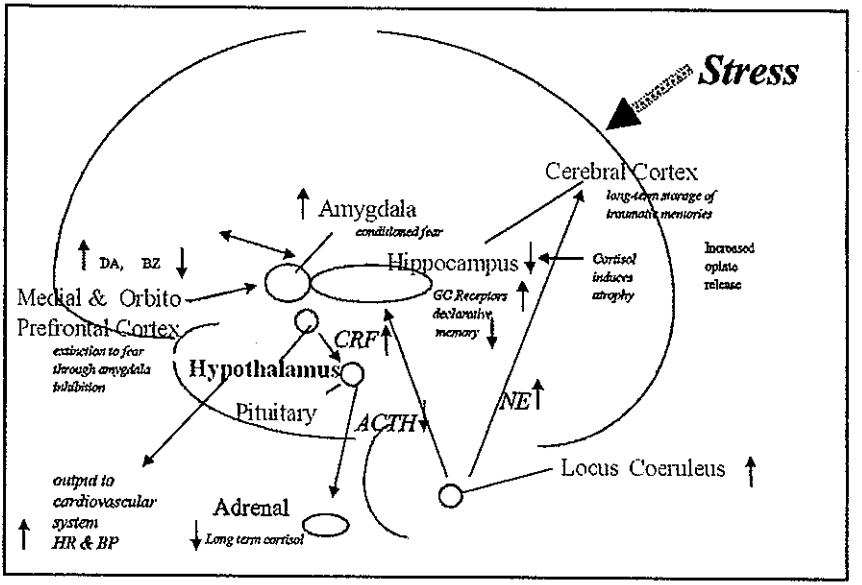
The model for the effects of traumatic stress on brain systems and structures involved in memory may have clinical relevance for psychotherapy of childhood abuse survivors. Psychotherapy may influence delayed recall of childhood abuse. Psychotherapy naturally involves the facilitation of recall through encouraging the investigation of feelings related to traumatic events. The psychotherapist may provide a supportive environment that allows the patient to experience strong emotions, which s/he may be afraid to experience outside of the therapeutic setting. If brain systems and structures that mediate memory are dysfunctional in patients with abuse-related PTSD, then recall of childhood abuse may only occur in the context of special situations, such as

psychotherapy sessions. Psychotherapy may therefore facilitate remembrance in ways other than providing false suggestions or promoting insertions of memories that never, in fact, took place. This may explain why some traumatic events are fully recalled for the first time during psychotherapy, which has fueled the controversy about whether these recalled events are true or false. The fact that traumatic events are recalled during therapy does not necessarily imply that they represent a false memory. Many therapists are nevertheless concerned about how to proceed with psychotherapy in light of the recent controversy concerning the engendering of false memories of abuse. They feel that if they ask directly about abuse, they may be accused of suggesting abuse events to their patients that did not, in fact, take place. Patients should be allowed to tell their own story, not the story of their therapists. Considering the evidence supporting the potential for suggestion to facilitate false memory illusions, it is important to avoid imposing one's own ideas as a therapist on the patient regarding a past history of abuse. Patients with PTSD may be even more susceptible to suggestion than normal persons. However, due to issues of shame and other reasons, they may not report abuse unless asked directly.

CONCLUDING COMMENTS

This article has presented a biological model for delayed recall of episodes of childhood abuse. The model is based on studies in animals and patients with abuse-related PTSD, and is therefore not generalizable to all situations, rather it is presented to stimulate thinking about possible neural mechanisms that may underlie delayed recall of childhood abuse. Evidence is reviewed showing that traumatic stress has lasting effects on brain chemical and structural systems involved in memory, emotion and the stress response, and that this may contribute to delayed recall of childhood abuse, was presented. Cortisol and norepinephrine released during stress can influence memory storage and retrieval, leading to either a weakening or a strengthening of memory traces. Long-term dysregulation of these systems occurs in abuse-related PTSD that may further influence memory retrieval and contribute to delayed recall effects (Figure 4). The hippocampus is particularly sensitive to stress, and stress-induced hippocampal damage may lead to deficits in declarative memory as well as impairments in the ability of the hippocampus to integrate memory elements at the time of retrieval. Hippocampal damage in PTSD may be associated with impairment in normal memory retrieval, leading to delayed recall in abuse survivors with PTSD. Another brain area affected by stress is the medial prefrontal cortex. Functional imaging studies show dysfunction in this area during presentation of traumatic reminders. Medial prefrontal cortical inhibition of amygdala responsiveness is felt to

FIGURE 4. Lasting Effects of Childhood Abuse on the Brain, Showing Long-Term Dysregulation of Norepinephrine and Cortisol Systems, and Vulnerable Areas of the Hippocampus, Amygdala, and Medial Prefrontal Cortex That Are Affected by Abuse



underlie extinction to fear responding. Dysfunction of this area in abuse-related PTSD may lead to problems modulating emotion that result in an avoidance of reminders of the trauma or associations to memories connected to the trauma. Memories of abuse may be "walled off" until they erupt into consciousness at a later time point. Prefrontal cortical dysfunction may also contribute to problems with source amnesia or memory distortion that may actually be more common in abuse-related PTSD, and that further complicate the story related to delayed recall of childhood abuse.

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